Exhibit 1

UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

FORM 10-K

(Mark One)

☒ ANNUAL REPORT PURSUANT TO SECTION 13 OR 15(d) OF THE SECURITIES EXCHANGE ACT OF 1934

For the fiscal year ended **September 30, 2023**

☐ TRANSITION REPORT PURSUANT TO SECTION 13 OR 15(d) OF THE SECURITIES EXCHANGE ACT OF 1934

For the transition period from _____ to_____

Commission file number**: 001-37606**

ANAVEX LIFE SCIENCES CORP.

(Exact name of registrant as specified in its charter)

Nevada	98-0608404
(State or other jurisdiction of incorporation or organization)	(I.R.S. Employer Identification No.)
630 5th Avenue, 20th Floor, New York, NY USA	10111
(Address of principal executive offices)	(Zip Code)

Registrant's telephone number, including area code 1-844-689-3939

Securities registered under Section 12(b) of the Act:

Common Stock, $0.001 par value	AVXL	NASDAQ Stock Market LLC
Title of each class	Trading Symbol	Name of each exchange on which registered

Securities registered pursuant to Section 12(g) of the Act:
None
(Title of class)

Indicate by checkmark if the registrant is a well-known seasoned issuer, as defined in Rule 405 of the Securities Act.

Yes ☒ No ☐

Indicate by checkmark if the registrant is not required to file reports pursuant to Section 13 or 15(d) of the Act.

Yes ☐ No☒

Indicate by checkmark whether the registrant has (1) filed all reports required to be filed by Section 13 or 15(d) of the Securities Exchange Act of 1934 during the preceding 12 months (or for such shorter period that the registrant was required to file such reports), and (2) has been subject to such filing requirements for the past 90 days.

Yes ☒ No ☐

Indicate by check mark whether the registrant has submitted electronically every Interactive Data File required to be submitted pursuant to Rule 405 of Regulation S-T (§232.405 of this chapter) during the preceding 12 months (or for such shorter period that the registrant was required to submit and post such files).

Yes ☒No ☐

Indicate by checkmark whether the registrant is a large accelerated filer, an accelerated filer, a non-accelerated filer, a smaller reporting company, or an emerging growth company. See the definitions of "large accelerated filer," "accelerated filer", "smaller reporting company" and "emerging growth company" in Rule 12b-2 of the Exchange Act.

Large accelerated filer ☒ Accelerated filer ☐
Non-accelerated filer ☐ Smaller reporting company ☐
Emerging growth company☐

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act

☐

Indicate by check mark whether the registrant has filed a report on and attestation to its management's assessment of the effectiveness of its internal controls over financial reporting under Section 404(b) of the Sarbanes-Oxley Act (15 U.S.C. 7262(b)) by the registered public accounting firm that prepared or issued its audit report.

Yes ☒No ☐

If securities are registered pursuant to Section 12(b) of the Act, indicate by check mark whether the financial statements of the registrant included in the filing reflect the correction of an error to previously issued financial statements

☐

Indicate by check mark whether any of those error corrections are restatements that required a recovery analysis of incentive-based compensation received by any of the registrant's executive officers during the relevant recovery period pursuant to §240.10D-1(b).

☐

Indicate by check mark whether the registrant is a shell company (as defined in Rule 12b-2 of the Act).

Yes ☐ No ☒

State the aggregate market value of the voting and non-voting common equity held by non-affiliates computed by reference to the price at which the common equity was last sold, or the average bid and asked price of such common equity, as of the last business day of the registrant's most recently completed second fiscal quarter: $667 million based on a price of $8.57 per share, being the closing price of the registrant's common stock on March 31, 2023.

Indicate the number of shares outstanding of each of the registrant's classes of common stock, as of the latest practicable date: 82,086,511 issued and outstanding as of November 24, 2023.

DOCUMENTS INCORPORATED BY REFERENCE

None.

TABLE OF CONTENTS

PART I	6
ITEM 1. BUSINESS	6
ITEM 1A. RISK FACTORS	32
ITEM 1B. UNRESOLVED STAFF COMMENTS	60
ITEM 2. PROPERTIES	60
ITEM 3. LEGAL PROCEEDINGS	60
ITEM 4. MINE SAFETY DISCLOSURES	61
PART II	61
ITEM 5. MARKET FOR REGISTRANT'S COMMON EQUITY, RELATED STOCKHOLDER MATTERS AND ISSUER PURCHASES OF EQUITY SECURITIES	61
ITEM 7 MANAGEMENT'S DISCUSSION AND ANALYSIS OF FINANCIAL CONDITION AND RESULTS OF OPERATION	61
ITEM 7A QUANTITATIVE AND QUALITATIVE DISCLOSURES ABOUT MARKET RISK	67
ITEM 8. FINANCIAL STATEMENTS AND SUPPLEMENTARY DATA	F-1
ITEM 9. CHANGES IN AND DISAGREEMENTS WITH ACCOUNTANTS ON ACCOUNTING AND FINANCIAL MATTERS	69
ITEM 9A. CONTROLS AND PROCEDURES	69
ITEM 9B OTHER INFORMATION	70
ITEM 9C DISCLOSURE REGARDING FOREIGN JURISDICTIONS THAT PREVENT INSPECTIONS	70
PART III	70
ITEM 10 DIRECTORS, EXECUTIVE OFFICERS AND CORPORATE GOVERNANCE	70
ITEM 11. EXECUTIVE COMPENSATION	74
ITEM 12. SECURITY OWNERSHIP OF CERTAIN BENEFICIAL OWNERS AND MANAGEMENT AND RELATED STOCKHOLDER MATTERS.	82
ITEM 13. CERTAIN RELATIONSHIPS AND RELATED TRANSACTIONS, AND DIRECTOR INDEPENDENCE	86
ITEM 14. PRINCIPAL ACCOUNTING FEES AND SERVICES	86
PART IV	88
ITEM 15. EXHIBITS, FINANCIAL STATEMENT SCHEDULES	88
ITEM 16. FORM 10-K SUMMARY	89

Forward Looking Statements.

This Annual Report on Form 10-K includes forward-looking statements. All statements other than statements of historical facts contained in this Annual Report on Form 10-K, including statements regarding our anticipated future clinical and regulatory milestone events, future financial position, business strategy and plans and objectives of management for future operations, are forward-looking statements. The words "believe," "may," "estimate," "continue," "anticipate," "intend," "expect," "should," "forecast," "potential," "predict," "could," "would", "will", "suggest," "plan" and similar expressions, as they relate to us, are intended to identify forward-looking statements. Such forward-looking statements include, without limitation, statements regarding:

- volatility in our stock price and in the markets in general;
- our ability to successfully conduct preclinical studies and clinical trials for our product candidates;
- our ability to raise additional capital on favorable terms and the impact of such activities on our stockholders and stock price;
- our ability to generate any revenue or to continue as a going concern;
- our ability to execute our research and development plan on time and on budget;
- our product candidates' ability to demonstrate efficacy or an acceptable safety profile;
- our ability to obtain the support of qualified scientific collaborators;
- our ability, whether alone or with commercial partners, to successfully commercialize any of our product candidates that may be approved for sale;
- our ability to identify and obtain additional product candidates;
- our reliance on third parties in non-clinical studies and clinical trials;
- our ability to defend against product liability claims;
- our ability to safeguard against security breaches;
- our ability to obtain and maintain sufficient intellectual property protection for our product candidates;
- our ability to comply with our intellectual property licensing agreements;
- our ability to defend against claims of intellectual property infringement;
- our ability to comply with the maintenance requirements of the government patent agencies;
- our ability to protect our intellectual property rights throughout the world;
- competition;
- the anticipated start dates, durations and completion dates of our ongoing and future clinical trials;
- the anticipated designs of our future clinical trials;
- our ability to attract and retain qualified employees;
- the impact of Fast Track designation on receipt of actual FDA approval;
- our anticipated future regulatory submissions and our ability to receive regulatory approvals to develop and market our product candidates, including any orphan drug or Fast Track designations; and
- our anticipated future cash position and ability to obtain funding for our operations.

We have based these forward-looking statements largely on our current expectations and projections about future events, including the responses we expect from the U.S. Food and Drug Administration, ("FDA"), and other regulatory authorities and financial trends that we believe may affect our financial condition, results of operations, business strategy, preclinical studies and clinical trials, and financial needs. These forward-looking statements are subject to a number of risks, uncertainties and assumptions including, without limitation, the risks described in "Risk Factors" in Part I, Item 1A of this Annual Report on Form 10-K. These risks are not exhaustive. Other sections of this Annual Report on Form 10-K include additional factors which could adversely impact our business and financial performance. Moreover, we operate in a very competitive and rapidly changing environment. New risk factors emerge from time to time and it is not possible for our management to predict all risk factors, nor can we assess the impact of all factors on our business or the extent to which any factor, or combination of factors, may cause actual results to differ materially from those contained in any forward-looking statements. You should not rely upon forward-looking statements as predictions of future events. We cannot assure you that the events and circumstances reflected in the forward-looking statements will be achieved or occur and actual results could differ materially from those projected in the forward-looking statements. Except as required by applicable laws including the securities laws of the United States, we assume no obligation to update or supplement forward-looking statements.

As used in this Annual Report on Form 10-K, the terms "we," "us," "our,", "Company" and "Anavex" mean Anavex Life Sciences Corp., unless the context clearly requires otherwise.

PART I

ITEM 1. BUSINESS

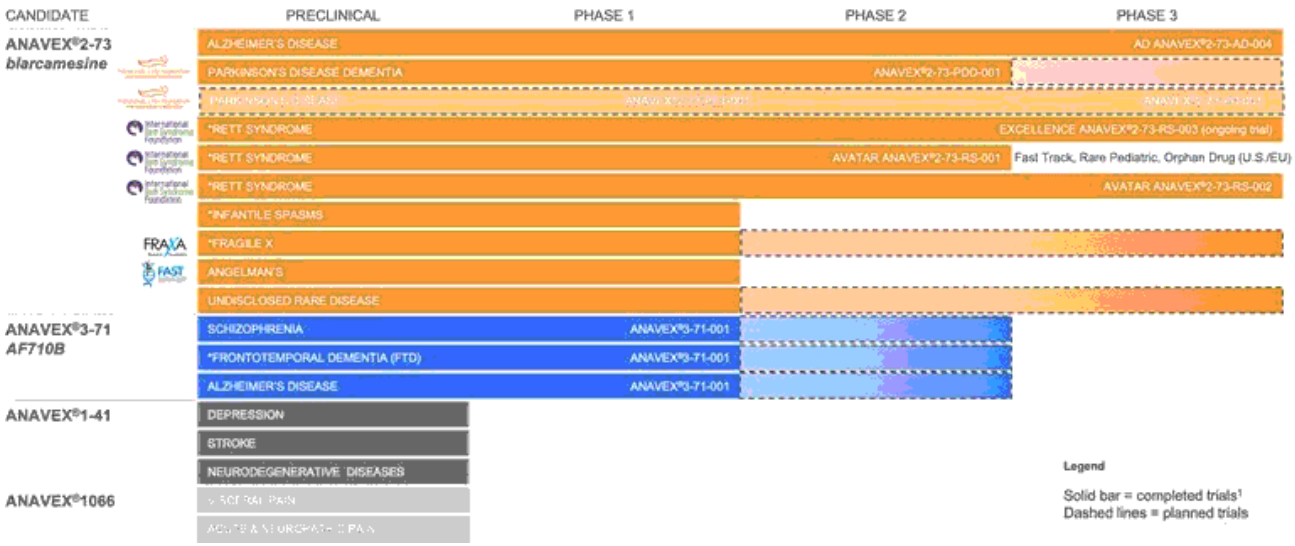
Overview and Strategy

Anavex Life Sciences Corp. is a clinical stage biopharmaceutical company engaged in the development of differentiated therapeutics by applying precision medicine to central nervous system ("CNS") diseases with high unmet need. We analyze genomic data from clinical trials to identify biomarkers, which we use in the analysis of our clinical trials.

Our lead product candidate, ANAVEX®2-73 (blarcamesine), is being developed to treat Alzheimer's disease, Parkinson's disease and potentially other central nervous system diseases, including rare diseases, such as Rett syndrome, a rare severe neurological monogenic disorder caused by mutations in the X-linked gene, methyl-CpG-binding protein 2 ("MECP2").

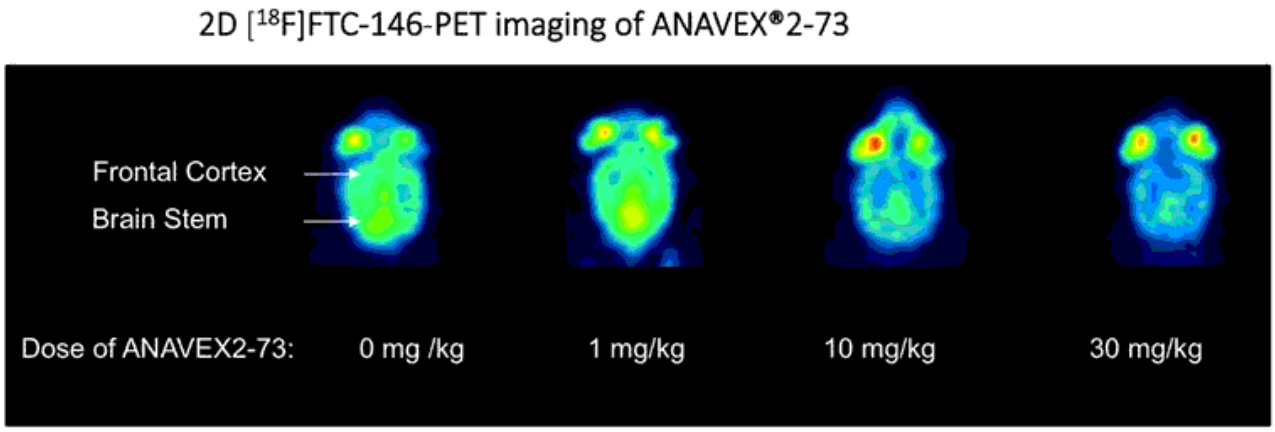
We currently have two core programs and two seed programs. Our core programs are at various stages of clinical and preclinical development, in neurodegenerative and neurodevelopmental diseases.

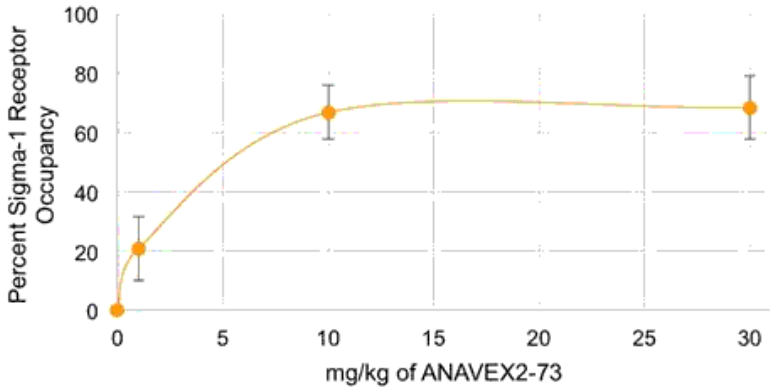
The following table summarizes key information about our programs:



** = Orphan Drug Designation by the FDA; 1. EXCELLENCE: Ongoing trial*

Anavex has a portfolio of compounds varying in sigma-1 receptor (SIGMAR1) binding activities. The SIGMAR1 gene encodes the SIGMAR1 protein, which is an intracellular chaperone protein with important roles in cellular communication. SIGMAR1 is also involved in transcriptional regulation at the nuclear envelope and restores homeostasis and stimulates recovery of cell function when activated. In order to validate the ability of our compounds to activate quantitatively the SIGMAR1, we performed, in collaboration with Stanford University, a quantitative Positron Emission Tomography (PET) imaging scan in mice, which demonstrated a dose-dependent ANAVEX®2-73 (blarcamesine) target engagement or receptor occupancy with SIGMAR1 in the brain.





Sigma-1 receptor target occupancy study with quantitative PET scan of ANAVEX®2-73

Source: Reyes S et al., Sci Rep. 2021 Aug 25;11(1):17150

Cellular Homeostasis

Many diseases are possibly directly caused by chronic homeostatic imbalances or cellular stress of brain cells. In pediatric diseases, such as Rett syndrome or infantile spasms, the chronic cellular stress is possibly caused by the presence of a constant genetic mutation. In neurodegenerative diseases, such as Alzheimer's and Parkinson's diseases, chronic cellular stress is possibly caused by age-correlated buildup of cellular insult and hence chronic cellular stress. Specifically, defects in homeostasis of protein or ribonucleic acid ("RNA") lead to the death of neurons and dysfunction of the nervous system. The spreading of protein aggregates resulting in a proteinopathy, a characteristic found in Alzheimer's and Parkinson's diseases that results from disorders of protein synthesis, trafficking, folding, processing or degradation in cells. The clearance of macromolecules in the brain is particularly susceptible to imbalances that result in aggregation and degeneration in nerve cells. For example, Alzheimer's disease pathology is characterized by the presence of amyloid plaques, and neurofibrillary tangles, which are aggregates of hyperphosphorylated Tau protein that are a marker of other diseases known as tauopathies as well as inflammation of microglia. With the SIGMAR1 activation through SIGMAR1 agonists like ANAVEX®2-73 (blarcamesine), our approach is to restore cellular balance (i.e. homeostasis). Therapies that correct defects in cellular homeostasis might have the potential to halt or delay neurodevelopmental and neurodegenerative disease progression.

ANAVEX®2-73 (blarcamesine)-specific Biomarkers

As part of some of our clinical trials, we have incorporated a genomic analysis to better understand potential populations for whom our clinical programs might benefit. In our clinical trials, a full genomic analysis of Alzheimer's disease patients treated with ANAVEX®2-73 (blarcamesine) has helped us identify actionable genetic variants. A significant impact of the genomic biomarkers SIGMAR1, the direct target of ANAVEX®2-73 (blarcamesine) and COMT, a gene involved in memory function, on the drug response level was identified, leading to an early ANAVEX®2-73 (blarcamesine)-specific biomarker hypothesis. We believe that *excluding* patients with SIGMAR1 identified biomarker variant (approximately 10%-20% of the population) in prospective studies would identify approximately 80%-90% patients that would display clinically significant improved functional and cognitive scores. The consistency between the identified DNA and RNA data related to ANAVEX®2-73 (blarcamesine), which are considered independent of Alzheimer's disease pathology, as well as multiple endpoints and time-points, provides support for the potential precision medicine clinical development of ANAVEX®2-73 (blarcamesine) by using genetic biomarkers identified within the trial population itself to target patients who are most likely to respond to ANAVEX®2-73 (blarcamesine) treatment. We may in the future utilize such an approach in Alzheimer's disease as well as indications like Parkinson's disease dementia or Rett syndrome in which ANAVEX®2-73 (blarcamesine) is currently being studied.

Clinical Trials Overview

Alzheimer's Disease

In November 2016, we completed a Phase 2a clinical trial, consisting of Part A and Part B, which lasted a total of 57 weeks, for ANAVEX®2-73 in mild-to-moderate Alzheimer's patients. This open-label randomized trial in Australia met both primary and secondary endpoints and was designed to assess the safety and exploratory efficacy of ANAVEX®2-73 in 32 patients. ANAVEX®2-73 targets sigma-1 and muscarinic receptors, which have been shown in preclinical studies to reduce stress levels in the brain believed to restore cellular homeostasis and to reverse the pathological hallmarks observed in Alzheimer's disease. In October 2017, we presented positive pharmacokinetic ("PK") and pharmacodynamic ("PD") data from the Phase 2a clinical trial, which established a concentration-effect relationship between ANAVEX®2-73 and trial measurements. These measures obtained from all patients who participated in the entire 57 weeks include exploratory cognitive and functional scores as well as biomarker signals of brain activity. Additionally, the clinical trial appeared to show that ANAVEX®2-73 activity was enhanced by its active metabolite (ANAVEX19-144), which also targets the SIGMAR1 receptor and has a half-life approximately twice as long as the parent molecule.

Two consecutive trial extensions for the Phase 2a trial have allowed participants who completed the 52-week Part B of the trial to continue taking ANAVEX®2-73, providing an opportunity to gather extended safety data for a cumulative time period of five years. In August 2020, patients completing these Phase 2a trial extensions were granted continued access to treatment with ANAVEX®2-73 through the Australian Government Department of Health - Therapeutic Goods Administration's compassionate use Special Access Scheme.

A larger Phase 2b/3 double-blind, placebo-controlled trial of ANAVEX®2-73 in Alzheimer's disease commenced in August 2018. The Phase 2b/3 trial enrolled 509 patients, which were treated with a convenient once-daily oral formulation of ANAVEX®2-73 for 48 weeks, randomized 1:1:1 to two different ANAVEX®2-73 doses or placebo. The trial took place at 52 sites across North America, Europe and Australia. Primary and secondary endpoints to assess safety and both cognitive and functional efficacy, were measured through the Alzheimer's Disease Assessment Scale - Cognitive Subscale test ("ADAS-Cog"), Alzheimer's Disease Cooperative Study - Activities of Daily Living ("ADCS-ADL") and Clinical Dementia Rating - Sum of Boxes for cognition and function ("CDR-SB"). In addition to the primary endpoints, the ANAVEX®2-73 Phase 2b/3 trial design incorporated pre-specified statistical analyses related to potential genomic precision medicine biomarkers previously identified in the ANAVEX®2-73 Phase 2a clinical trial. The trial was completed in mid-2022 and, in December 2022, the Company presented positive topline results from the Phase 2b/3 clinical trial.

ANAVEX®2-73 met the co-primary endpoints ADAS-Cog and ADCS-ADL and key secondary endpoint CDR-SB. ANAVEX®2-73 treatment slowed decline of cognition and function in patients with early Alzheimer's disease over 48 weeks. Patients treated with ANAVEX®2-73 had 1.84 times higher odds, or likelihood, to improve cognitively compared to placebo, with a ADAS-Cog score threshold change of -0.5 points or better [Odds Ratio = 1.84 (p = 0.015)]. At clinically significant levels of improvement in function (ADCS-ADL score threshold change of +3.5 points or better), patients treated with ANAVEX®2-73 had 2.67 times higher odds, or likelihood, to improve function compared to placebo [Odds Ratio = 2.67 (p = 0.0255)]. Additionally, treatment with ANAVEX®2-73 reduced cognitive decline at end of treatment, measured with the ADAS-Cog, as compared to placebo, by 45%, representing a treatment difference in mean score change of -1.85 points (p=0.033). Compared to placebo, ANAVEX®2-73 reduced clinical decline of cognition and function by 27% with mean score difference of -0.42 points (p=0.040) as measured by the CDR-SB. ANAVEX®2-73 was generally safe and well tolerated. All statistical analyses were performed by outside consultancy companies.

In September 2023, we provided additional data demonstrating that the clinical effect was complemented by two independent biomarkers. A significant reduction in pathological amyloid beta levels in plasma, as well as a significant slowing in the rate of pathological brain atrophy on MRI (Magnetic Resonance Imaging) scans. Validated biomarkers of amyloid beta pathology, plasma Aβ42/40 ratio increased significantly (P = 0.048), demonstrating strong anti-amyloid effects of ANAVEX®2-73 in Alzheimer's disease patients, while MRI revealed significant reduction in brain volume loss, including whole brain (P = 0.0005), comparing treatment to placebo.

Furthermore, all prespecified clinical endpoints were further analyzed using a mixed model for repeated measures (MMRM). Under the multiplicity control rule, a trial is successful in meeting the co-primary endpoints if the significance of each endpoint is $P < 0.05$, or if the significance of only one co-primary endpoint is $P < 0.025$. If only one primary endpoint is significant at an α level of 0.025, then the secondary endpoint will be evaluated at the same level of 0.025. The trial was successful, since the differences in the least-squares mean (LSM) change from baseline to 48 weeks between the ANAVEX®2-73 and placebo groups were −1.783 [95% CI, −3.314 to −0.251]; (P = 0.0226) for ADAS-Cog13, and −0.456 [95% CI, −0.831 to −0.080]; (P = 0.0175) for CDR-SB in patients with early Alzheimer's disease.

In the respective safety population, common treatment-emergent adverse events included dizziness, which was transient and mostly mild to moderate in severity, and occurred in 120 participants (35.8%) during titration and in 76 participants (25.2%) during maintenance with ANAVEX®2-73 and 10 (6.0%) during titration and 9 (5.6%) during maintenance with placebo.

A subsequent long-term open label extension study of ANAVEX®2-73, entitled the ATTENTION-AD trial was initiated for patients who have completed the 48-week Phase 2b/3 placebo-controlled trial referenced above. This trial extension for an additional 96 weeks is currently ongoing, and provides an opportunity to evaluate longer term safety and efficacy of ANAVEX®2-73 in persons with Alzheimer's disease.

Rett Syndrome

In February 2016, we presented positive preclinical data for ANAVEX®2-73 in Rett syndrome, a rare neurodevelopmental disease. The data demonstrated dose related and significant improvements in an array of behavioral and gait paradigms in a mouse model with an MECP2-null mutation that causes neurological symptoms that mimic Rett syndrome. The study was funded by the International Rett Syndrome Foundation ("Rettsyndrome.org"). In January 2017, we were awarded a financial grant from Rettsyndrome.org of a minimum of $0.6 million to cover some of the costs of a multicenter Phase 2 clinical trial of ANAVEX®2-73 for the treatment of Rett syndrome. This award was received in quarterly instalments which commenced during fiscal 2018.

In March 2019, we commenced the first Phase 2 clinical trial in a planned Rett syndrome program of ANAVEX®2-73 for the treatment of Rett syndrome. The clinical trials are being conducted in a range of patient age demographics and geographic regions, utilizing an oral liquid once-daily formulation of ANAVEX®2-73.

The first Phase 2 trial, (ANAVEX®2-73-RS-001), which took place in the United States, was completed in December 2020. This trial was a randomized double-blind, placebo-controlled safety, tolerability, PK and efficacy trial of oral liquid ANAVEX®2-73 formulation in 25 adult female patients with Rett syndrome over a 7-week treatment period including ANAVEX®2-73-specific genomic precision medicine biomarkers. The primary endpoint of the trial was safety. The dosing of 5 mg ANAVEX®2-73 was well-tolerated and demonstrated dose-proportional PK. All secondary efficacy endpoints of the trial showed statistically significant and clinically meaningful response in the Rett Syndrome Behaviour Questionnaire ("RSBQ") response, when compared to placebo, in the intent to treat ("ITT") cohort (all participants, p = 0.011). 66.7% of ANAVEX®2-73 treated subjects showed a statistically significant improvement in RSBQ response as compared to 10% of the subjects on placebo in the ITT cohort (all participants, p = 0.011). ANAVEX®2-73 treatment resulted in a sustained improvement in Clinical Global Impression Improvement (CGI-I) response throughout the 7-week clinical trial, when compared to placebo in the ITT cohort (all participants, p = 0.014). Consistent with previous ANAVEX®2-73 clinical trials, patients carrying the common form of the SIGMAR1 gene treated with ANAVEX®2-73 experienced stronger improvements in the prespecified efficacy endpoints.

The second, international trial of ANAVEX®2-73 for the treatment of Rett syndrome, called the AVATAR trial, commenced in June 2019. This trial took place in Australia and the United Kingdom using a higher dose than the U.S. based Phase 2 trial for Rett syndrome. The trial was a Phase 3 randomized, double-blind, placebo-controlled trial to evaluate the safety and efficacy of ANAVEX®2-73 in 33 adult patients over a 7-week treatment period including ANAVEX®2-73 specific precision medicine biomarkers. Based upon the input from the successful U.S. Phase 2 Rett syndrome trial (ANAVEX®2-73-RS-001), we updated the endpoints for the AVATAR trial (ANAVEX®2-73-RS-002) to appropriately assess the clinically meaningful outcome following International Conference on Harmonization (ICH) guidelines. These updates were approved by the respective regulatory authorities in the U.K. and in Australia, respectively, where the AVATAR trial was conducted.

The data from the AVATAR trial was released in February 2022. The clinical trial met all primary and secondary efficacy and safety endpoints, with consistent improvements in primary efficacy endpoint, RSBQ response (p = 0.037), and secondary efficacy endpoints, ADAMS (p = 0.010) and CGI-I (p = 0.037) response. Efficacy endpoints demonstrated statistically significant and clinically meaningful reductions in Rett syndrome symptoms. Convenient once daily oral liquid doses of up to 30 mg of ANAVEX®2-73 were also well tolerated with good medication compliance. All patients who participated in the trial were eligible to receive ANAVEX®2-73 under a voluntary open label extension protocol.

In July 2020, we commenced the third trial of ANAVEX®2-73 for the treatment of Rett syndrome, called the EXCELLENCE trial. This Phase 2/3 trial in pediatric patients with Rett syndrome includes trial sites in Australia, the United Kingdom and Canada, and will evaluate the safety and efficacy of ANAVEX®2-73 in approximately 84 pediatric patients, aged 5 to 18, over a 12-week treatment period incorporating ANAVEX®2-73 specific precision medicine biomarkers. This trial completed enrollment in February 2023, exceeding the original enrollment target. In June 2023, this trial completed dosing of all participants, and topline results are expected in the second half of 2023. All patients who participate in the trial will be eligible to receive ANAVEX®2-73 under a voluntary open label extension protocol, which is currently ongoing.

Parkinson's Disease

In September 2016, we presented positive preclinical data for ANAVEX®2-73 in an animal model of Parkinson's disease, which demonstrated significant improvements on behavioral, histopathological, and neuroinflammatory endpoints. The study was funded by the Michael J. Fox Foundation. Additional data announced in October 2017 indicated that ANAVEX®2-73 induced robust neurorestoration in experimental Parkinsonism. We believe the encouraging results we have gathered in this preclinical model, coupled with the favorable profile of this product candidate in the Alzheimer's disease trial, support the notion that ANAVEX®2-73 has the potential to treat Parkinson's disease dementia.

In October 2020, we completed a double-blind, randomized, placebo-controlled proof-of-concept Phase 2 trial with ANAVEX®2-73 in Parkinson's disease dementia in Spain and Australia, to study the effect of the compound on both the cognitive and motor impairment of Parkinson's disease. The Phase 2 trial enrolled approximately 132 patients for 14 weeks, randomized 1:1:1 to two different ANAVEX®2-73 doses, 30 mg and 50 mg, or placebo. The ANAVEX®2-73 Phase 2 Parkinson's disease dementia trial design incorporated genomic precision medicine biomarkers identified in the ANAVEX®2-73 Phase 2a Alzheimer's disease trial.

The trial demonstrated that ANAVEX®2-73 was safe and well tolerated in oral doses up to 50 mg once daily. The results showed clinically meaningful, dose-dependent, and statistically significant improvements in the Cognitive Drug Research ("CDR") computerized assessment system analysis. Treatment with ANAVEX®2-73 also resulted in clinically meaningful improvements as measured by the global composite score of Parkinson's disease symptom severity, MDS-Unified Parkinson's Disease Rating Scale ("MDS-UPDRS") total score on top of standard of care including dopaminergic therapy, levodopa and other anti-PD medications after 14 weeks of treatment, suggesting ANAVEX®2-73's potential capability of slowing and reversing symptoms that progress in Parkinson's disease. In addition, the trial confirmed the precision medicine approach of targeting SIGMAR1 as a genetic biomarker in response to ANAVEX®2-73 may result in improved clinical outcomes.

A 48-week Open Label Extension ("OLE") ANAVEX2-73-PDD-EP-001 Phase 2 trial was offered to participants after completion of the double-blind placebo-controlled ANAVEX2-73-PDD-001 Phase 2 trial discussed above. The OLE trial assessed safety, tolerability and efficacy, measuring among others, MDS-Unified Parkinson's Disease Rating Scale Parts I, II, III, REM Sleep Behavior Disorder Screening Questionnaire (RBDSQ), Clinical Global Impression - Improvement (CGI-I), as well as cognitive efficacy endpoint Montreal Cognitive Assessment (MoCA) over a 48-week period.

In March 2023, we reported the preliminary ANAVEX2-73-PDD-EP-001 OLE trial data, which demonstrated longitudinal beneficial effects of ANAVEX®2-73 on the prespecified primary and secondary objectives. Preliminary analysis reveals that ANAVEX®2-73 was found to be generally safe and well tolerated; and safety findings in this trial were consistent with the known safety profile of ANAVEX®2-73. In respect to efficacy, across all efficacy endpoints, patients performed better while on ANAVEX®2-73. While all patients were on drug holiday due to COVID-19 between the DB EOT and the OLE Baseline, the respective efficacy endpoints, including the MDS-UPDRS Part II + III and CGI-I, measured at the end of trial of the double-blind study (DB EOT) and the OLE Baseline, were worsening, as expected in a progressive disease like Parkinson's. However, when patients resumed daily oral ANAVEX®2-73 treatment, a consistent improvement was observed during the extension phase from OLE Baseline through OLE Week 24, and OLE Week 48, respectively. These results are consistent with the pattern observed for all efficacy measures in the extension phase. The two endpoints, MDS-UPDRS Part II + III and CGI-I measured in this study are the planned primary and key secondary endpoints in our forthcoming pivotal 6-month Parkinson's disease study.

In January 2021, we were awarded a research grant of $1.0 million from The Michael J. Fox Foundation for Parkinson's Research to develop ANAVEX®2-73 for the treatment of Parkinson's disease. The award will explore utilization of PET imaging biomarkers to enable measurement of target engagement and pathway activation of the SIGMAR1 with clinically relevant doses, including in people with Parkinson's disease.

Schizophrenia, Frontotemporal Dementia and Alzheimer's disease

In July 2020, we commenced the First-in-Human Phase 1 clinical trial of ANAVEX®3-71. ANAVEX®3-71 was previously granted orphan drug designation for the treatment of Frontotemporal Dementia ("FTD") by the FDA. ANAVEX®3-71 is an orally administered small molecule targeting sigma-1 and M1 muscarinic receptors that is designed to be beneficial for neurodegenerative diseases. In preclinical studies, ANAVEX®3-71 demonstrated disease-modifying activity against the major hallmarks of Alzheimer's disease in transgenic (3xTg-AD) mice, including cognitive deficits, amyloid and tau pathologies, as well as beneficial effects on mitochondrial dysfunction and neuroinflammation.

The Phase 1 clinical trial was a prospective double-blind, randomized, placebo-controlled trial in Australia. A total of 36 healthy male and female subjects were included. Single escalating doses of ANAVEX®3-71 were administered in order to evaluate the safety, tolerability, and PK of ANAVEX®3-71 and the effects of food and gender on its PK in healthy volunteers.

The trial met its primary and secondary endpoints of safety, with no serious adverse events ("SAEs") or dose-limiting toxicities observed. ANAVEX®3-71 was well tolerated in all cohorts receiving ANAVEX®3-71 in single doses ranging from 5 mg to 200 mg daily with no SAEs and no significant lab abnormalities in any subject. In the trial, ANAVEX®3-71 exhibited linear PK. Its pharmacokinetics was also dose proportional for doses up to 160 mg. Gender had no effect on the PK of the drug and food had no effect on the bioavailability of ANAVEX®3-71. The trial also met the secondary objective of characterizing the effect of ANAVEX®3-71 on electrocardiogram ("ECG") parameters. There were no clinically significant ECG parameters throughout the trial. Participant QTcF measures were normal across all dose groups with no difference between ANAVEX®3-71 and placebo.

In October 2023 a peer-reviewed publication in the journal Neurobiology of Aging, titled "Early treatment with an M1 and sigma-1 receptor agonist prevents cognitive decline in a transgenic rat model displaying Alzheimer-like amyloid pathology", featured the orally available small molecule ANAVEX®3-71 *(AF710B*). The preclinical study described the potential disease-modifying properties of ANAVEX®3-71 on Alzheimer's disease pathology as a possible drug candidate for a potential once daily oral preventive strategy for Alzheimer's disease.

Based on these results, and ANAVEX®3-71's pre-clinical profile, we intend to advance ANAVEX®3-71 into a biomarker-driven clinical development dementia program for the treatment of schizophrenia, FTD and Alzheimer's disease, evaluating longitudinal effect of treatment with ANAVEX®3-71. We believe the results of these clinical trials and preclinical study could serve as a basis for advancing into respective registration trials in the U.S.

Our Pipeline

Our research and development pipeline includes ANAVEX®2-73 currently in three different clinical trial indications, and several other compounds in different stages of clinical and pre-clinical development.

Our proprietary SIGMACEPTOR™ Discovery Platform produced small molecule drug candidates with unique modes of action, based on our understanding of sigma receptors. Sigma receptors may be targets for therapeutics to combat many human diseases, both of neurodegenerative nature, including Alzheimer's disease, as well as of neurodevelopmental nature, like Rett syndrome. When bound by the appropriate ligands, sigma receptors influence the functioning of multiple biochemical signals that are involved in the pathogenesis (origin or development) of disease. Multiple viruses including SARS-CoV-2 (COVID-19) induce cellular stress by intrinsic mitochondrial apoptosis and other related cellular processes, in order to ensure survival and replication. Hence, it is possible that SIGMAR1 could play a role in modulating the cellular response to viral infection and ameliorate pathogenesis.

Compounds that have been subjects of our research include the following:

ANAVEX®2-73 *(blarcamesine)*

We believe ANAVEX®2-73 may offer a disease-modifying approach in neurodegenerative and neurodevelopmental diseases by activation of SIGMAR1. ANAVEX®2-73 is being developed in an oral liquid once-daily formulation for rare diseases such as Rett syndrome as well as an oral once-daily capsule formulation for diseases such as Alzheimer's disease.

In Rett syndrome, administration of ANAVEX®2-73 in liquid form resulted in both significant and dose related improvements in an array of behavioral paradigms in the MECP2 HET Rett syndrome disease model. In addition, in a further experiment sponsored by Rettsyndrome.org, ANAVEX®2-73 was evaluated in automatic visual response and respiration tests in 7-month old mice, an age at which advanced pathology is evident. Vehicle-treated MECP2 mice demonstrated fewer automatic visual responses than wild-type mice. Treatment with ANAVEX®2-73 for four weeks significantly increased the automatic visual response in the MECP2 Rett syndrome disease mice. Additionally, chronic oral dosing daily for 6.5 weeks of ANAVEX®2-73 starting at ~5.5 weeks of age was conducted in the MECP2 HET Rett syndrome disease mouse model assessed the different aspects of muscular coordination, balance, motor learning and muscular strengths, some of the core deficits observed in Rett syndrome. Administration of ANAVEX®2-73 resulted in both significant and dose related improvements in an array of these behavioral paradigms in the MECP2 HET Rett syndrome disease model.

In May 2016 and June 2016, the FDA granted Orphan Drug Designation to ANAVEX®2-73 for the treatment of Rett syndrome and infantile spasms, respectively. In November 2019, the FDA granted to ANAVEX®2-73 the Rare Pediatric Disease (RPD) designation for the treatment of Rett syndrome. The RPD designation is intended to encourage the development of treatments for rare pediatric diseases.

Further, in February 2020, the FDA granted Fast Track designation for the ANAVEX®2-73 clinical development program for the treatment of Rett syndrome. The FDA Fast Track program is designed to facilitate and expedite the development and review of new drugs to address unmet medical needs in the treatment of serious and life-threatening conditions.

For Parkinson's disease, data demonstrates significant improvements and restoration of function in a disease-modifying animal model of Parkinson's disease. Significant improvements were seen on all measures tested: behavioral, histopathological, and neuroinflammatory endpoints. In October 2020, we completed a double-blind, randomized, placebo-controlled proof-of-concept Phase 2 trial with ANAVEX®2-73 in Parkinson's disease dementia, to study the effect of the compound on both the cognitive and motor impairment of Parkinson's disease. The Phase 2 trial enrolled approximately 132 patients for 14 weeks, randomized 1:1:1 to two different ANAVEX®2-73 doses, 30mg and 50mg, or placebo. The ANAVEX®2-73 Phase 2 Parkinson's disease dementia trial design incorporated genomic precision medicine biomarkers identified in the ANAVEX®2-73 Phase 2a Alzheimer's disease trial.

The trial demonstrated that ANAVEX®2-73 was safe and well tolerated in oral doses up to 50mg once daily. The results showed clinically meaningful, dose-dependent, and statistically significant improvements in the CDR computerized assessment system analysis. We anticipate conducting further clinical trials of ANAVEX®2-73 in Parkinson's disease dementia after submitting the results of the trial to the FDA to obtain regulatory guidance.

In Alzheimer's disease animal models, ANAVEX®2-73 has shown pharmacological, histological and behavioral evidence as a potential neuroprotective, anti-amnesic, anti-convulsive and anti-depressive therapeutic agent, due to its potent affinity to SIGMAR1 and moderate affinities to M1-4 type muscarinic receptors. In addition, ANAVEX®2-73 has shown a potential dual mechanism which may impact amyloid, tau pathology and inflammation. In a transgenic Alzheimer's disease animal model Tg2576, ANAVEX®2-73 induced a statistically significant neuroprotective effect against the development of oxidative stress in the mouse brain, as well as significantly increased the expression of functional and synaptic plasticity markers that is apparently amyloid-beta independent. It also statistically alleviated the learning and memory deficits developed over time in the animals, regardless of sex, both in terms of spatial working memory and long-term spatial reference memory.

Based on the results of pre-clinical testing, we initiated and completed a Phase 1 single ascending dose (SAD) clinical trial of ANAVEX®2-73. In this Phase 1 SAD trial, the maximum tolerated single dose was defined per protocol as 55-60 mg. This dose is above the equivalent dose shown to have positive effects in mouse models of Alzheimer's disease. There were no significant changes in laboratory or ECG parameters. ANAVEX®2-73 was well tolerated below the 55-60 mg dose with only mild adverse events in some subjects. Observed adverse events at doses above the maximum tolerated single dose included headache and dizziness, which were moderate in severity and reversible. These side effects are often seen with drugs that target CNS conditions, including Alzheimer's disease.

In November 2016, we completed a Phase 2a clinical trial for ANAVEX®2-73, for the treatment of Alzheimer's disease. The open-label randomized trial was designed to assess the safety and exploratory efficacy of ANAVEX®2-73 in 32 patients with mild-to-moderate Alzheimer's disease. The Phase 2a trial met both primary and secondary objectives of the trial.

In July 2018, we presented the results of a genomic DNA and RNA evaluation of the participants in the Phase 2a clinical trial. More than 33,000 genes were analyzed using unbiased, data driven, machine learning, artificial intelligence (AI) system for analyzing DNA and RNA data in patients treated with ANAVEX®2-73. The analysis identified genetic variants that impacted response to ANAVEX®2-73, among them variants related to the SIGMAR1, the target for ANAVEX®2-73. Results showed that trial participants with the common SIGMAR1 wild type gene variant, which is estimated to be about 80% of the population worldwide, demonstrated improved cognitive (MMSE) and functional (ADCS-ADL) scores. The results from this evaluation supported the continued evaluation of genomic information in subsequent clinical trials, since these signatures can now be applied to neurological indications tested in future clinical trials with ANAVEX®2-73 including Alzheimer's disease, Parkinson's disease dementia and Rett syndrome.

ANAVEX®2-73 data met prerequisite information in order to progress into a Phase 2b/3 placebo-controlled trial. On July 2, 2018, the Human Research Ethics Committee in Australia approved the initiation of our Phase 2b/3, double-blind, randomized, placebo-controlled 48-week safety and efficacy trial of ANAVEX®2-73 for the treatment of early Alzheimer's disease. Clinical trial sites in Canada, the United Kingdom, the Netherlands and Germany were also added. This Phase 2b/3 trial design incorporates inclusion of genomic precision medicine biomarkers identified in the ANAVEX®2-73 Phase 2a trial.

We believe preclinical data from our studies also supports further research into the use of ANAVEX®2-73 as a potential platform drug for other neurodegenerative diseases beyond Alzheimer's disease, Parkinson's disease or Rett syndrome, more specifically, epilepsy, infantile spasms, Fragile X syndrome, Angelman syndrome, multiple sclerosis, and, more recently, tuberous sclerosis complex (TSC). ANAVEX®2-73 demonstrated significant improvements in all of these indications in the respective preclinical animal models.

In a preclinical study sponsored by the Foundation for Angelman Syndrome, ANAVEX®2-73 was assessed in a mouse model for the development of audiogenic seizures. The results indicated that ANAVEX®2-73 administration significantly reduced audiogenic-induced seizures in mice. In a study sponsored by FRAXA Research Foundation regarding Fragile X syndrome, data demonstrated that ANAVEX®2-73 restored hippocampal brain-derived neurotrophic factor (BDNF) expression to normal levels. BDNF under-expression has been observed in many neurodevelopmental and neurodegenerative pathologies. BDNF signaling promotes maturation of both excitatory and inhibitory synapses. ANAVEX®2-73 normalization of BDNF expression could be a contributing factor for the positive preclinical data observed in both neurodevelopmental and neurodegenerative disorders like Angelman and Fragile X syndromes.

In addition, preclinical data to-date also indicates that ANAVEX®2-73 has the potential to demonstrate protective effects of mitochondrial enzyme complexes during pathological conditions, which, if impaired, may play a role in the pathogenesis of neurodegenerative and neurodevelopmental diseases.

In addition, preclinical data on ANAVEX®2-73 related to multiple sclerosis indicates that ANAVEX®2-73 may promote remyelination in multiple sclerosis disease. Further, our data also demonstrates that ANAVEX®2-73 has the potential to provide protection for oligodendrocytes ("OL's") and oligodendrocyte precursor cells ("OPC's"), as well as central nervous system neurons in addition to helping repair by increasing OPC proliferation and maturation in tissue culture.

In March 2018, we presented preclinical data of ANAVEX®2-73 in a genetic mouse model of tuberous sclerosis complex ("TSC"). TSC is a rare genetic disorder characterized by the growth of numerous benign tumors in many parts of the body with a high incidence of seizures. The preclinical data demonstrated that treatment with ANAVEX®2-73 significantly increased survival and reduced seizures in those mice.

ANAVEX®3-71

ANAVEX®3-71 is a clinical drug candidate with a novel mechanism of action via SIGMAR1 activation and M1 muscarinic allosteric modulation, which has been shown to enhance neuroprotection and cognition in Alzheimer's disease models. ANAVEX®3-71 is a CNS-penetrable potential disease-modifying treatment for cognitive impairments. We believe it is effective in very small doses against the major Alzheimer's hallmarks in transgenic (3xTg-AD) mice, including cognitive deficits, amyloid and tau pathologies, and also has beneficial effects on inflammation and mitochondrial dysfunctions. ANAVEX®3-71 indicates extensive therapeutic advantages in Alzheimer's and other protein-aggregation-related diseases given its ability to enhance neuroprotection and cognition via SIGMAR1 activation and M1 muscarinic allosteric modulation.

SIGNATURES

Pursuant to the requirements of Section 13 or 15(d) of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned thereunto duly authorized.

Date: November 27, 2023

ANAVEX LIFE SCIENCES CORP.

By: */s/ Christopher Missling, PhD*
Name: Christopher Missling, PhD
Title: Chief Executive Officer (Principal Executive Officer)

Pursuant to the requirements of the Securities Exchange Act of 1934, this report has been signed below by the following persons on behalf of the registrant and in the capacities and on the dates indicated.

Signatures	Title(s)	Date
/s/ Christopher Missling, PhD Christopher Missling, PhD	Chief Executive Officer (Principal Executive Officer) and Director	November 27, 2023
/s/ Sandra Boenisch Sandra Boenisch, CPA, CGA	Principal Financial Officer and Treasurer (Principal Accounting Officer)	November 27, 2023
/s/ Jiong Ma, PhD Jiong Ma, PhD	Director, Chair	November 27, 2023
/s/ Athanasios Skarpelos Athanasios Skarpelos	Director	November 27, 2023
/s/ Claus van der Velden, PhD Claus van der Velden, PhD	Director	November 27, 2023
/s/ Steffen Thomas, PhD Steffen Thomas, PhD	Director	November 27, 2023
/s/ Peter Donhauser, D.O. Peter Donhauser, D.O.	Director	November 27, 2023

Exhibit 21.1

SUBSIDIARIES OF THE REGISTRANT

Name of Subsidiary	Jurisdiction of Incorporation or Organization
Anavex Australia Pty Limited	Australia
Anavex Germany GmbH	Germany
Anavex Canada Ltd.	Ontario, Canada

EXHIBIT 23.1

CONSENT OF INDEPENDENT REGISTERED PUBLIC ACCOUNTING FIRM

We have issued our reports dated November 27, 2023, with respect to the consolidated financial statements and internal control over financial reporting included in the Annual Report of Anavex Life Sciences Corp. on Form 10-K for the year ended September 30, 2023. We consent to the incorporation by reference of said reports in the Registration Statements of Anavex Life Sciences Corp. on Forms S-3 (No. 333-218292, and No. 333-259788) and Forms S-8 (No. 333-219934, No. 333-255166 and No. 333-265537).

/s/ GRANT THORNTON LLP

Melville, New York
November 27, 2023

EXHIBIT 23.2

Consent of Independent Registered Public Accounting Firm

Anavex Life Sciences Corp.
New York, New York

We hereby consent to the incorporation by reference in the Registration Statements on Form S-3 (No. 333-218292 and No. 333-259788) and Form S-8 (No. 333-219934, No. 333-255166 and No. 333-265537) of Anavex Life Sciences Corp. and subsidiaries of our report dated November 24, 2021, relating to the consolidated financial statements of Anavex Life Sciences Corp. and subsidiaries, which appears in this Annual Report on Form 10-K.

/s/ BDO USA, P.C.

New York, New York
November 27, 2023

EXHIBIT 31.1

CERTIFICATION

I, Christopher Missling, certify that:

1. I have reviewed this Annual Report on Form 10-K of Anavex Life Sciences Corp.;

2. Based on my knowledge, this report does not contain any untrue statement of a material fact or omit to state a material fact necessary to make the statements made, in light of the circumstances under which such statements were made, not misleading with respect to the period covered by this report;

3. Based on my knowledge, the financial statements, and other financial information included in this report, fairly present in all material respects the financial condition, results of operations and cash flows of the registrant as of, and for, the periods presented in this report;

4. The registrant's other certifying officer(s) and I are responsible for establishing and maintaining disclosure controls and procedures (as defined in Exchange Act Rules 13a-15(e) and 15d-15(e)) and internal control over financial reporting (as defined in Exchange Act Rules 13a-15(f) and 15d-15(f)) for the registrant and have:

   a) Designed such disclosure controls and procedures, or caused such disclosure controls and procedures to be designed under our supervision, to ensure that material information relating to the registrant, including its consolidated subsidiaries, is made known to us by others within those entities, particularly during the period in which this report is being prepared;

   b) Designed such internal control over financial reporting, or caused such internal control over financial reporting to be designed under our supervision, to provide reasonable assurance regarding the reliability of financial reporting and the preparation of financial statements for external purposes in accordance with generally accepted accounting principles;

   c) Evaluated the effectiveness of the registrant's disclosure controls and procedures and presented in this report our conclusions about the effectiveness of the disclosure controls and procedures, as of the end of the period covered by this report based on such evaluation; and

   d) Disclosed in this report any change in the registrant's internal control over financial reporting that occurred during the registrant's most recent fiscal quarter (the registrant's fourth fiscal quarter in the case of an annual report) that has materially affected, or is reasonably likely to materially affect, the registrant's internal control over financial reporting; and

5. The registrant's other certifying officer(s) and I have disclosed, based on our most recent evaluation of internal control over financial reporting, to the registrant's auditors and the audit committee of the registrant's board of directors (or persons performing the equivalent functions):

   a) All significant deficiencies and material weaknesses in the design or operation of internal control over financial reporting which are reasonably likely to adversely affect the registrant's ability to record, process, summarize and report financial information; and

   b) Any fraud, whether or not material, that involves management or other employees who have a significant role in the registrant's internal control over financial reporting.

Date: November 27, 2023

/s/ Christopher Missling

Christopher Missling, PhD
Chief Executive Officer, President, Secretary
(Principal Executive Officer)